Crossed Leg Palsy

With Report of a Recurrent Case

ISIDORE I. WEISS, M.D., *Stockton*

SUMMARY

A form of peroneal palsy may be caused by crossing the legs. Two physical factors—pressure and tension — are the basic causes, although other factors may be contributory. Direct pressure is applied by the bones of the two legs, compressing the peroneal nerve between them at its superficial part near the head and neck of the fibula.

The palsy may be overlooked as an integral part of a widespread disorder so that careful evaluation and observation of the patient's habits are required. Detection becomes especially difficult when the palsy is bilateral, for then the lesion by virtue of its symmetry blends more readily with associated polyneuritis. A case of recurrent peroneal palsy due to crossing the legs in a prolonged postoperative convalescence is reported in detail.

CROSSING the legs when seated is extremely common and yet the foot drop it causes is by comparison very rare. In some circumstances, when the subject in this position exposes the peroneal nerve to prolonged pressure, crossed leg paralysis may result. In the normal subject the nerve is protected by soft parts except for the superficial inch or two near the head and neck of the fibula. There it may be injured by plaster casts, adhesive tape, or prolonged pressure on the operating room table or in delivery room stirrups, and peroneal paralysis may result.

Textbooks discuss these causes of peroneal palsy but even recent ones do little more than mention crossing the legs as a cause. Nielsen,[7] for example, states, "The nerve is usually injured in its exposed position as it winds around the head of the fibula by crossing the knees," but, like other texts, his does not describe the background of these cases. For the details we must turn to the literature, especially the contributions by Woltman,[9] who reported the first comprehensive study (27 civilians, in 1929), and the latest by Nagler and Rangell[6] (eight soldiers, in 1947). Laird and Mueller[5] called it "bombardier's palsy" but it was found also in other soldiers who were not bombardiers.[6] As Woltman found a farmer, stenographer, auctioneer, professional golfer, jeweler, and a blacksmith among the afflicted, occupation cannot be considered a predisposing factor. The lesion is also found in the unemployed, and their very unemployment may be a contributory factor, for ". . . man is not always occupied, and when he is not, he enjoys the unique privilege of crossing his knees with the result that the peroneal nerve is often wedged between the head of the ipsilateral fibula and the external condyle of the heterolateral femur and the heterolateral patella."[9] It may occur even when one falls asleep with the legs crossed.[4]

CONVALESCENTS SUSCEPTIBLE

Peroneal palsy may be found in convalescents. Paget[8] in 1876 reported it in convalescence from typhoid. Bing[1] reported a case in a patient convalescent from nasal diphtheria who sat a long time in a physician's waiting room with his knees crossed. Most of the 27 cases reported by Woltman were in patients convalescing in a hospital. Sitting idly, the convalescent is likely to keep his legs crossed for unusually long periods. In a case observed by the author the lesion developed first in a patient soon after operation, with recurrence five months later in the same convalescence. In discussing the role of an operation, Woltman[9] said, "In surgical cases it appeared more frequently before operations than after, regardless of the type of operation and of the presence of infection."

A paralysis so easily prevented should not recur, and it seldom does. The following case is reported because only one other in which there was recurrence is recorded in the literature—the sixth of the eight cases reported by Nagler and Rangell.[6] It is not stated how long after recovery the paralysis recurred in that case, but in the case observed by the author it was five months.

ETIOLOGY

Pressure alone may cause the paralysis when the nerve is in direct contact with the bones of both legs, but only when certain predisposing factors exist. Woltman's[9] patients were in the fourth or fifth decade of life, were inactive because of illness, had lost considerable weight, and had other contributory factors causing neuritis. In contrast, Nagler and Rangell[6] reported most of their patients were in the 30-40 age group, were actively engaged (and activity rather than inactivity played an important part), and were generally healthy (only 25 per cent had lost weight). Probably more pertinent to the problem is the observation by Nagler and Rangell that when pyknic, short-legged persons cross their legs there is as much as six inches between the neck of the fibula (with the peroneal nerve around it) and the point at which the legs are crossed. Thus the

peroneal nerve is not compressed between them. But persons of the daddy-long-legs type can and do cross their legs in such a way that the point of intersection is higher and the peroneal nerve subjected to pressure. Their patients were tall (average height 71½ inches), thin, and long-legged. In squatting or kneeling at work they stretched the peroneal nerve, causing ischemia. Direct pressure on the nerve between the bones of the two legs, when they sat with legs crossed, always caused ischemia.

WEIGHT LOSS A PREDISPOSING FACTOR

Tall, thin persons, however, are not alone in predisposition to the lesion. Palsy of this origin occurs in others as well, when severe loss of weight so alters the body contour that the peroneal nerve, possibly already debilitated and more sensitive to injury as corollary of the factors that caused the weight loss, may be compressed when the legs are crossed. Close inspection of subjects seated with legs crossed shows that those in whom paralysis develops literally wind one leg around the other so that the nerve is stretched when the crossed leg hugs the straight leg. Then the position becomes pathogenic, for the "hugging" and "winding" cause, respectively, pressure and tension which are simultaneous and prolonged, both injurious to the nerve. Nagler and Rangell pointed out that kneeling and squatting create tension, and that sitting with legs crossed exposes the nerve to direct pressure. They did not, however, emphasize that both the stretching and pressure can occur at the same time in the single act of sitting with legs crossed, and that this becomes possible in a person of any skeletal type who has lost enough weight to permit proximation of the bones of the two legs.

CASE REPORT

The patient, a white male 51 years of age, complained of weakness in the right foot and numbness in the ankle. In walking the right foot slapped the floor and had to be lifted higher than the left to prevent its scraping. The patient could not dorsiflex the right foot nor extend the toes. Five months previously identical paralysis had occurred during convalescence soon after gastrectomy but had cleared up after two weeks of vitamin injections.

The patient, who was 69 inches in height, had lost 35 pounds in weight postoperatively and had regained only 10. Appetite was poor and there had been diarrhea intermittently for several months. The buttocks had wasted away, the thighs had poor muscle tone, and the muscle bellies in front of and below the knees were wasted and hollow. Although the paralysis was unilateral, the two limbs were symmetrical. Sphincter tone and control were intact. There were no sensory changes in the saddle area. Position and vibration sensation were normal. The patient could stand on the left leg alone to test position sensation, but not on the right. Pin prick and light touch sensation were impaired in the lower anterolateral aspect of the right leg, and more so on the dorsum of the foot and ankle. No sensory changes were found in the left leg. Pressing the peroneal nerve against the fibula produced a painful sensation in the left leg and foot but not in the right. The deep reflexes, hyperactive at the knees, were barely elicited at the ankles in the Oppenheim position, and then only with reinforcements.

Although there was complaint of tingling and numbness in the fingers and hands, there were no objective findings in the upper limbs. The pupils were slightly unequal and scarcely reacted to light. The left disc showed questionable pallor with a clearly defined nasal margin; the right was normal. No abnormalities were elicited in other cranial tests.

Occupational stress was not a factor. For many years the patient had operated a complicated paper manufacturing machine, a block long, servicing it as a machinist. Ill health had never kept him away from work. He had returned to work three months postoperatively but found he was too weak. When the foot drop recurred he stayed indoors to avoid uneven ground.

The diagnosis was polyneuritis caused by prolonged disturbances in alimentation and made worse by a postoperative "dumping syndrome" and diarrhea which further depleted low vitamin reserves. But the advanced isolated paralysis was not in harmony with the symmetry of the polyneuritis. It was difficult to explain this until it was noted that when the patient crossed his legs, he literally "wrapped" the right leg around the left, covering it from knee to ankle. It was a life-long habit, he said, and he always placed the right leg on the left. Preoperatively he had been unable to keep this position for an extended time, but with the postoperative weight loss he could rest one leg comfortably and snugly against the other for hours at a time. The patient recalled that just before the paralysis recurred he had sat this way for four hours in a poker game. Moreover, as his legs had been wedged firmly between the chair and the table, it is probable that the prolonged pressure on the nerve was greater than it might otherwise have been.

The patient was told to have his wife and friends remind him of it whenever he crossed his legs, since he continued to do it even after being cautioned against it. Thiamin chloride, 50 mg., was given thrice daily, and nicotinic acid for its vasodilator effect; also massage with frequent immersion of the affected foot in a bucket of hot water. The foot was rested against a box when the patient was reclining, in order to minimize stretching of the weakened muscles. The patient gained a pound a week. First the numbness left; then the gait improved. In two months the hollows below the knees began to fill in, and the numbness of the fingers was felt only in cool weather. In three months, recovery was complete and the patient returned to work.

DIFFERENTIAL DIAGNOSIS

So selective a paralysis should not be, but sometimes is, a diagnostic problem. For example, in one of the cases reported by Nagler and Rangell[6] the patient had extensive acute polyradiculoneuritis of Guillain-Barré with lower motor neuron disease, and the peroneal palsy was much more profound than the rest of the weakness. The peroneal palsy could easily have been diagnosed incorrectly as an integral part of the polyradiculoneuritis. In the author's case, such an error was made for a while. The error may become serious, for in a case reported by Eaton,[3] it "almost led to needless laminectomy." Nagler and Rangell made a similar observation: "In one case suspicion of disease of the spinal cord led to extensive investigation and almost to needless laminectomy, while in two others functional diagnoses were made and psychotherapy given for long periods prior to accurate diagnosis." Accurate diagnosis is even more difficult when the syndrome is bilateral, for then it blends with the symmetry of polyneuritis. As Dunning[2] reported one such case and Woltman found bilateral peroneal palsy in five cases in a series of 27, the bilateral lesion cannot be

considered a rarity. A history of venereal disease in the presence of such a paralysis and of obscure extensive neurological signs may tempt one to consider the lesion syphilitic, but spinal fluid examination clarifies the issue. In 25 per cent of Nagler and Rangell's[6] cases, physicians had tentatively considered hysteria when no other apparent cause could be found.

State Hospital.

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